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A Study of the Volume and Specific Gravity of Organs.[1]

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THE three linear dimensions in which the size of an organ examined at autopsy is recorded give to the reader a rather indefinite idea as to the actual size of the specimen. Viscera are irregular. The expressions "the organ is large," "fairly large," "voluminous," "larger than its fellow," "contracted," "splenic tumor," etc., are inaccurate and unscientific.

In order to determine and accurately register the size of any viscus, the following plan, employed at some institutions for registering the volume of the brain, should be adopted: Each organ as it is removed is submerged in a vessel filled with water to a level at which an overflow is provided. The water displaced overflows into a container

[1] Read by invitation.

graduated in cubic centimeters; the amount so obtained represents the volume or displacement of the organ in cubic centimeters. The organs are weighed in grams; the weight in grams, divided by the displacement in cubic centimeters, equals the specific gravity.

The heart is submerged opened or unopened. A "voluminous" emphysematous lung is pressed beneath the surface of the water by a rod thrust into the bronchus. It has been ascertained that no water enters the lungs—no bubbles appear because the contained air cannot be displaced. The liver, spleen, kidneys, or a tumor may be similarly measured. The record is thereby supplied with definite facts by which it is possible to appreciate the size of an emphysematous lung as compared with its atelectatic mate. The displacement in cubic centimeters constitutes a record, conveying an idea not obtainable from linear measurements even when the weight also is given.

Dr. L. Vervaeck, of Belgium, determined the specific gravity of organs and published his results in 1901. The method he used requires two weighings: one in and one out of water. His tabulations were based on the general clinical diagnosis of the case and not on the pathological condition observed in each organ. The specific gravity of the lungs was not determined.

In order to determine the practical value of the methods suggested I examined the organs from one hundred autopsies made at the Philadelphia Hospital.[1]

HEART.—While the average specific gravity of hearts manifesting no evident abnormality was 1029, individual apparently normal hearts varied from 939 to 1152. It is not likely that healthy heart muscle varies in specific gravity to the extent indicated. Any marked deviation from the normal specific gravity, in a heart macroscopically normal, indicates that a histological study is necessary. In cloudy swelling—8 cases—the average specific gravity was found distinctly lowered, namely, to 1004. This conforms to observations made during the study, namely, that parenchymatous change lowers the specific gravity of the affected organ. In so-called chronic myocarditis, including general atrophy (20 cases), the average specific

[1] The notes of the cases studied may be found in the Philadelphia Hospital records, 1906, xxi.

gravity was 1008; lowest, 833; highest, 1088. Hypertrophied and dilated hearts (31 cases) yielded an average specific gravity of 1037; lowest, 969; highest, 1114. In simple hypertrophy (13 cases) the average specific gravity was 1032; lowest, 975; highest, 1128. Comparing hypertrophy with hypertrophy and dilatation combined, the previous observations seemed again to hold true. In dilatation, failure of nutrition and parenchymatous degeneration, the specific gravity was, as a rule, lower than in simple hypertrophy. The same general tendency obtains in chronic dilatation and fatty degeneration. Here with even greater parenchymatous change (12 cases) the average specific gravity is still lower, namely, 1025; lowest, 843; highest, 1214. Fatty infiltration presents a contrasting picture. Here with an intact musculature the specific gravity remained high, the average of 5 cases being 1061; lowest, 1000; highest, 1151.

KIDNEY.—The average specific gravity of the normal kidney (only 8 such available) was 1098; lowest, 1000; highest, 1190. In acute diffuse nephritis (20 kidneys) the average specific gravity was lowered to 1072.

In chronic parenchymatous nephritis (103 kidneys), commonly a diffuse lesion, the average specific gravity was further lowered, 1049. In one case the right kidney had a specific gravity of 1400. The left kidney was less granular; accordingly its specific gravity was only 1200. In chronic interstitial nephritis (58 kidneys) the average specific gravity, 1053, was higher than in chronic parenchymatous nephritis. As illustrating the influence of morbid processes in the displacement—size—of the organ, a comparison of the average volumes of the kidneys is interesting and suggestive.

Average volume, chronic interstitial nephritis (58 cases)	150 c.c.
Average volume, apparently normal kidneys (8 cases)	160 c.c.
Average volume, chronic parenchymatous nephritis (103 cases)	166 c.c.
Average volume, acute diffuse nephritis (20 cases)	185 c.c.
Average volume, acute diffuse nephritis and congestion (8 cases)	186 c.c.

While parenchymatous change seems to lower the specific gravity of the organ, the formation of fibrous tissue, on the other hand, raises it. This is perhaps best indicated by examination of the kidneys in chronic interstitial nephritis. Assuming that the kidneys of the smallest vol-

ume have undergone the most interstitial change—are most fibrous—the following comparisons may be made:

8 kidneys chronic interstitial nephritis, vol. 100 c.c. or less, av. sp. gr. 1257.
42 kidneys chronic interstitial nephritis,[1] vol. 100 to 200 c.c., av. sp. gr. 1025.
8 kidneys chronic interstitial nephritis,[2] vol. over 200 c.c. av. sp. gr. 994.

The organ becomes more dense because it contracts. The increased specific gravity contributed by fibrous-tissue formation is not demonstrable in every case, be it heart, liver, or kidney, because fibrous or interstitial processes are rarely dissociated from parenchymatous change. Where fibrosis is most marked, as in the group of small kidneys in chronic interstitial nephritis, the consequent increase of specific gravity is best illustrated.

LIVER.—In organs not the seat of any macroscopically evident lesion (11 cases) the average specific gravity was 1057; lowest, 1029; highest, 1088. The specific gravity is lowest in fatty infiltration of the liver (21 cases); average, 1028; lowest, 720; highest, 1098. In cloudy swelling, parenchymatous degeneration (12 cases), the average specific gravity was 1055; lowest, 1025; highest, 1086. In atrophic cirrhosis (7 cases) the average specific gravity was 1056; lowest, 1029; highest, 1069. In congestion amounting to red atrophy, average specific gravity was 1077; lowest, 973; highest, 1100.

SPLEEN.—Except in miliary tuberculosis the average specific gravity of the spleen is highest in chronic splenitis (28 cases), 1139, succeeded in order by the following: Acute splenitis (19 cases), 1110; congestion (17 cases), 1108; apparently normal (33 cases), 1043; acute suppurative splenitis (2 cases), 1040; amyloid (3 cases), 1027.

LUNGS.—As regards the lungs this method offers a more perfect mode of comparison of size of the two organs than can be obtained in any other way. In support of this statement the following cases may be cited:

CASE 38.[3]—Patient aged twenty-six years. Right lung: weight, 460; volume, 850; specific gravity, 541; chronic caseous tuberculosis. Left lung: weight, 780; volume, 770; specific gravity, 1013; chronic caseous tuberculosis and atelectasis of lower lobe.

[1] Probably less fibrous. [2] Probably least fibrous.
[3] Weight is given in grams and displacement, or volume, in cubic centimeters.

It is clear in this case that the right was the functionating lung; less weight, greater displacement, lower specific gravity.

CASE 39.—Patient, aged thirty-five years. Right lung: weight, 590; volume, 810; specific gravity, 728; edema and congestion; tuberculosis of lower lobe, hence higher specific gravity. Left lung: weight, 400; volume, 640; specific gravity, 625; edema and congestion.

CASE 53.—Patient, aged fifty-seven years. Right lung: weight, 560; volume, 580; specific gravity, 965; emphysema, congestion, healed tuberculosis. Left lung: weight, 190; volume, 180; specific gravity, 1055; atelectasis.

No description could give as adequate an idea of the conditions in this case as the figures quoted.

CASE 76.—Patient, aged twenty-four years. Right lung: weight, 460; volume, 670; specific gravity, 686; acute miliary tuberculosis. Left lung: weight, 460; volume, 610; specific gravity, 754; acute miliary tuberculosis.

The two lungs weighed the same; the right lung was larger; the left should have weighed less; the specific gravity of the left was the higher. From the figures alone it is proper to conclude that the left lung was the more involved.

CASE 88.—Patient, aged forty-six years. Right lung: weight, 660; volume, 1200; specific gravity, 500; emphysema. Left lung: weight, 940; volume, 1300; specific gravity, 723; lobar pneumonia involving part of upper lobe; the remainder of the organ emphysematous.

CASE 108.—Patient, aged fifty years. Right lung: weight, 540; volume, 1150; specific gravity, 469; the organ apparently normal, crepitating throughout. Left lung: weight, 1790; volume, 1780; specific gravity, 1005; lobar pneumonia; nowhere crepitant.

These few cases are sufficient to illustrate what is already known, namely, that conditions like fibrosis, atelectasis, and pneumonia increase the specific gravity of the lung. Comparison of weights, volumes, and specific gravity of the two lungs gives an approximate idea of the amount of functionating tissue present in each.

CONCLUSIONS.—A statistical study of the organs of 100 consecutive autopsies seems to indicate:

1. Parenchymatous degeneration lowers the specific gravity of organs proportionately to the degree of parenchymatous change.

2. Fibrotic change, while diminishing the volume of the organ, also raises its specific gravity proportionately to the amount of fibrosis.

3. Although useful in systematic studies of all organs, the specific gravity records are most striking in pulmonary affections.

March 12, 1908.

Note on the Occurrence of a Ciliate (*Opalinopsis nucleolobata, n. s.*) in the Liver of a Mammal (*Canis latrans*).

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The following record seems to the writers worthy of publication, because, so far as they are aware, there is but one other case mentioned in medical literature in which a ciliate was noted as a parasite of the mammalian liver, and because, provided the identification of the organism here dealt with as of the family of *Opalinidæ* be correct, it is the first time in which it has been found that any member of this family has been parasitic in a mammal, the various species being known only as parasites of worms and other invertebrates, and of frogs and toads.

In stained sections[1] of the liver of a prairie wolf, *canis latrans*,[2] these ciliates were discovered in large numbers. Unfortunately, the writers are forced to depend entirely upon preserved material, as no idea of their occurrence was had prior to their discovery in the finished histological preparations. This fact materially limits the study, as much of the examination of such specimens is necessarily or preferably to be carried out upon the fresh and living protozoa.

The coyote had been in the Zoölogical Gardens for about two years, but was a poor inbred specimen, was never on exhibition, and was ordered killed on April 3, 1907. The autopsy was performed very shortly after death and the material for microscopic examina-

[1] University of Pennsylvania Path. Hist., No. 2199.
[2] Philadelphia Zoölogical Garden Laboratory, 1048.

tion at once fixed in formaldehyde solution. There existed a hypostatic congestion of both lungs, and a slight grade of general visceral fibrosis, an interstitial nephritis being especially developed. In various places in the liver there were indefinitely outlined areas, varying from 15 to 30 millimeters in diameter, which upon the surface of the organ formed slightly convex prominences, somewhat paler than the rest of the hepatic substance and a trifle softer, superficially suggesting the appearances of small abscesses. In section, these swelled out above the rest of the cut surface and seemed to consist of irregularly shaped liver lobules with blotches of a brownish or yellowish-brown pigment in and among them. The alimentary canal was grossly normal, and no part was saved for microscopic examination.

In the histological preparations of the liver there were no notable alterations of the general structure (beyond a slight perilobular cirrhosis) save in connection with these areas. In the latter the blood-vessels were irregularly dilated, at places the tissue approaching the appearance of an angioma, this affecting especially the intralobular capillaries here and there, and there was a small amount of hemic pigmentary deposit. Here and there in these nodes were patches of hepatic cells presenting a fine vacuolation, converting the protoplasm into a fine reticulum, but without involvement or change in the nuclei (probably edema of the cells rather than fatty change). Between the liver cells and often definitely within the dilated blood channels of these nodes the ciliates were found, sometimes widely separated from each other or again in numbers in the tissue of a single field of the microscope. In the vicinity of the infusoria, and about them, there was often a minor infiltration of the tissue by polynuclear and rather large mononuclear leukocytes; but there was in no instance any definite encapsulation of the organisms. The gall-ducts showed no invasion by the parasites, the epithelial lining of these channels being quite normal, although in places there appeared a slight increase of the peribiliary connective tissue in the sections examined.

The organisms are spheroid to short ovoid in outline, the largest ones attaining a long diameter of 0.035 mm. (exclusive of the ciliary border); the smaller recognized individuals often being less than half of this measurement. In case of many of the parasites no ciliated border can be made out, this being particularly true of small

examples and individuals about which the cellular elements of the organ (hepatic cells, red blood corpuscles and leukocytes) are closely packed. None are seen with partial ciliation. Typically, the organisms are holotrichous, the entire border being thickly and uniformly set with rather coarse, more or less matted ciliæ, without any appreciable local differentiation at any position. The existence of cuticular striation is uncertain; it is not in the least evident in the great majority of examples, but there were very faint suggestions of a longitudinal striation noted in a few of the parasites (this point would have been clearer, doubtless, in the fresh state, and must be held in doubt). Beneath the ciliary surface there is a sharply defined and fairly thick, somewhat refractile cell wall taking a slight eosin tint in the hematoxylin and eosin preparations. The cytoplasm, staining in the same combination a faint eosin hue, is finely granular and without clear differentiation into endosarc and ectosarc. No trace of a mouth, pharyngeal depression, anal orifice, or vacuoles are recognizable, and the substance is entirely free from the coarse granules which are common as ingesta in most ciliates. The nucleus is very variable in its appearance. No micronuclei are recognized in examination of hundreds of examples (but the known difficulty of demonstrating the micronucleus of infusoria save in the fresh unstained specimen should be recalled and the failure to detect this body in our material is not to be regarded as certain evidence of its absence). Ordinarily the nucleus is relatively large, often occupying, especially in small individuals, quite as much of the cell as does the nucleus of a small lymphocyte. In the smaller specimens the nucleus is apt to be simple, round or oval, taking on the hematoxylin hue deeply. In the larger ones the nucleus is a large multilobulate mass, the lobules commonly clumped together so that their relations are not clear. A few instances in which the nucleus shows a circular or horseshoe shape, with the nodules appearing as swellings upon the band, and a few others with elongated, irregularly band-like nuclei, indicate to the minds of the writers that the parasites are not multinucleate, as in the genus *Opalina*, but that the nuclear lobulations all belong to one nuclear mass. Either as a stage of vital existence or as the result of regressive changes (more probably the former), nuclei are to be found broken into thick cord-like fragments,

reminding one of coarse chromatic elements in mitosis, but certainly not of the appearance of mitotic figures as found in infusoria (such nuclei, too, are clearly to be regarded as macronuclei, whereas mitosis in the infusoria involves the micronucleus). Division of the nucleus is apparently a direct one, and the division of the cell takes place by a simple hour-glass constriction following the nuclear division. The writers have also met with examples in which there are clearly separate nuclei or parts of nuclei, but after careful consideration are disposed to believe the picture artificial, due to section of the nucleus in such manner that some of the lobular divisions are separated from the major mass. So, too, we would interpret the occasional examples of non-nucleated ciliates in the sections on the assumption that they are parts of large examples which have been separated by the plane of section from the larger part of the cell which bears the nucleus. There were met relatively few examples enclosed in a double wall, but such may be accepted as instances of encystment of the parasites. The large number of specimens about which the ciliary border cannot be detected are in part evidently the result of obscuration of the ciliæ by the close packing of other elements about the infusoria, and in part probably to poor fixation (this idea is upheld by the fact that the ciliæ are by far best seen in sections made from thin blocks of tissue early selected and fixed for histological study, while the ciliation is less frequent and more poorly shown in material later taken from larger masses of the liver coarsely preserved); doubtless, too, in some instances the ciliæ have been retracted prior to encystment of the parasites.

It seems very improbable that in material thus prepared there could have been lost with uniformity all appearance of mouth, pharynx, contractile vacuoles, and similar organelles; and the writers, therefore feel justified in referring the organisms to the family *Opalinina*, which is characterized by the absence of these parts. The absence of coarse fragments from the cell, as are commonly ingested by infusoria, is confirmatory of this view; the organisms probably obtaining their nutrition and carrying on excretion entirely through the cuticle. Primarily, from the occasional appearance of separateness of the nuclei above referred to, and from the apparent absence of micronuclei, the writers believed the specimen to belong to the genus

Opalina; but more careful consideration of certain nuclei, and in instances where, probably from artefact, the nucleus is being extruded from the organism, has reversed this view and justified the idea of a single multilobulate nucleus. The characteristics determined coincide with those of the genus *Opalinopsis*, Foettinger,[1] 1881. It is to be noted, however, that none of our specimens exhibit the reticulate type of nucleus (nuclear substance as an anastomosing network occupying much of the cell) which Foettinger described in the species upon which the genus was based (*Opalinopsis sepiolæ*, *Opalinopsis elegans*, and *Opalinopsis octopi*), which were encountered in the liver of several sepiolæ and of an octopus. In the living animals, too, Foettinger was able to make out the existence of a number of small non-contractile vacuoles. If such existed in our material in the living state they may well have been so reduced in fixation as to have become inapparent. Careful comparison of material with Foettinger's text and description makes it certain that the specimens in hand are not specifically identical with any of the known species of opalinopsis, and the writers would offer the examples here described as a new species, for which they would suggest the name *Opalinopsis nucleolobata*. The determined features of the species in synopsis are: *Spherical or short ovate holotrichous infusoria, without oral or anal orifices, without pharyngeal depression; with no differentiation of the rather shaggy ciliæ at any position; apparently without any form of vacuole; micronucleus doubtful or absent; nucleus large, single, multilobate (simple in early forms), occasionally in band and beaded form and sometimes showing as coarse cord-like fragments; no definite differentiation between ectoplasm and endoplasm; size up to* 0.035 *mm. in greater diameter; cuticular striations uncertain.*

The only other instance known to either of the writers at present in which a ciliate has been found as a parasite in the mammalian liver is recorded in *Transactions of the Pathological Society of London*, 1899; *Paramœcium coli*, an occasional parasite of the human large intestine, having been met in the liver of a man who had died of malignant disease of the stomach. The living parasites were found in the cheesy contents of numerous small cysts connected with the

[1] Arch. de Biologie, 1881, ii, 354.

gall-ducts. Apparently the parasites had reached the liver by way of the common bile duct from the intestine, setting up an irritation which occasioned the formation of the cysts.

In the case above detailed it is quite uncertain how the parasites gained access to the liver of the coyote and what was the source. Opalinopsis as described by Foettinger is thus far known as a parasite of the liver in sepiola and octopus; perhaps the species encountered has its natural habitat also in the liver of some low type, and by preference sought this organ after being introduced into the economy by the alimentary tract. Occasionally canine animals devour frogs and toads or even lower forms of animal life; and, at least, this may be thought of as a probable source until the organism is discovered in its proper host. *March* 12, 1908.

Comparative Study of Noma.

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Noma is of interest on account of its comparative rarity and of the diverse opinions expressed by different observers in regard to its etiology. The condition becomes of added interest when found in the lower animals, and especially in a wild animal.

Various causes for this disease have been advanced. The older writers thought that the condition was due to a thrombosis or to trophic changes. These theories have been dismissed because the disease is not confined to the course of any one bloodvessel or nerve. The probability of its being of an infectious nature is now looked upon as certain.

The findings of the various observers may be roughly grouped under various heads. Thus:

(*a*) Moser[1] in three cases found protozoa.

(*b*) Walsh[2] and Levi and Sailor[3] found, culturally, the Bacillus diphtheriæ; Goepp,[4] the Bacillus diphtheriæ associated with the

Staphylococcus albus and the Streptococcus pyogenes; Petruschky,[5] the Bacillus diphtheriæ accompanied by vibrios and spirilla; and Bishop,[6] the Bacillus diphtheriæ both culturally and in the tissues.

(*c*) Ranke[7] found, both culturally and histologically, streptococci resembling those already described by Koch which occur in progressive tissue necrosis of field mice.

(*d*) Guizette[8] and Babés and Zambilovici[9] found fusiform-shaped bacilli both culturally and in the tissues, while Rosenberger[10] and Weaver and Tunnicliff[11] obtained in smears the Bacillus fusiformis accompanied by a spirillum similar to the organisms which Vincent[12] had previously noted in the necrotic tissues of hospital gangrene, a disease analogous to noma.

We now come to a large group of observers, many of recent date, whose findings, while similar in many respects, differ in the minuter details.

This group is composed of those who have found histologically, and in some cases culturally, bacillary microörganisms forming thread or filament-like processes, and in some cases spirillum forms.

As early as 1883, Lingard and Batt[13] described bacilli found in rapidly advancing necrotic disease in the mouths of cattle.

Later, Lingard[14] found, in necrosis of the mouth in man, monkeys, and calves, and in gangrenous pneumonia in horses, a bacillus forming long threads.

Grawitz[15] found bacilli forming long threads which were Gram positive.

Bartels[16] observed bacilli, threads, and cocci.

Schimmelbusch[17] noted rounded bacilli forming long threads in the central part of the gangrenous area.

Foote[18] reported the presence of long bacilli joining end to end in long strings.

Blumer and McFarlane[19] demonstrated a leptothrix forming short bacilli and long threads.

Perthes,[20] Krahn,[21] and Brüning[22] observed bacilli forming threads and spirillum forms (Perthes calling this organism a streptothrix).

Schmorl[23] found long thread-like organisms resembling the Bacillus necroseos.

Finally, Jensen[24] and others, viz., Bang, Kitt, McFadyean, etc.,

have noted the Bacillus necroseos in this condition in lower animals such as cattle, sheep, kangaroos, monkeys, and even birds.

Herrman,[25] in summing up the literature on noma, endeavors to simplify the different findings noted by advancing the theory that the spirillum of Vincent is simply a stage of development of the Bacillus fusiformis, and that this latter organism and the streptothrix of Perthes are identical with the Spirillum sputigenum and the Spirochete dentium which Miller found in the normal mouth as saprophytes. Herman further states that he considers the Spirochete dentium as only a stage of development of the Spirillum sputigenum, thus claiming (as previously held by Krahn) that noma is probably caused by the saprophyte Spirillum sputigenum assuming under favorable conditions a parasitic role. He would call this organism the Spirochete of necrosis. He still further states that he considers the Bacillus necroseos of lower animals to be closely related to this Spirochete necroseos.

All the observers who have studied the necrotic tissues histologically, have noted that in the superficial areas of advanced necrosis there was a polymicrobic infection, while the nearer they approached the healthy tissue the fewer became the microörganisms until along the zone between the healthy and necrotic tissues each found the particular microörganisms which he described in almost pure culture.

Notable among the reports of the various observers is that of Perthes, who found histologically along the line of demarcation rod- and spindle-shaped microörganisms forming long thread or filament-like processes and spirillum forms. The nearer the examination approached the healthy tissue the less frequently were found the threads, until along the immediate border of the necrotic tissue and growing into the healthy structures were demonstrated fine spirillum-like processes in great numbers. These rods, threads, and spirillum forms he considers to be different stages of development of a single microörganism, which he thinks is the same as the bacillary and thread-like organisms found by others in man and animals, the spirillum forms probably remaining hidden on account of their poor staining properties. Krahn and Brüning have corroborated his findings, Brüning further noting the penetration of the bloodvessel walls in the necrotic areas by these bacilli.

These bacilli, fibers, and spirillum forms were found to be Gram negative.

In regard to the clinical side of noma, it has been noted by several observers that it started as an ulcerative stomatitis; in fact, being an advanced stage of this condition or a gangrenous stomatitis. The primary lesion is generally a gingivitis beginning about the teeth and advancing to the grosser lesion of noma with all its characteristics.

The case which we would present to-night is one of noma or gangrenous stomatitis occurring in a wild animal, a Rhesus Macaque monkey at the Zoological Gardens of Philadelphia.

The subject being a wild animal, there is naturally not much of a clinical history.

This monkey had not been sent to the exhibition cages, but was still held in quarantine for further observation, when attention was called to a purplish discoloration limited to a small area upon the lower lip. This looked exactly like an ordinary bruise such as the animal might receive in handling or in the small cage. In twenty-four hours this area had increased greatly in size, practically involving the whole lower lip, which was greatly swollen, bluish black in color, and showed a distinct line of demarcation. Upon closer examination the mucous membrane of the gum was found to be ulcerated, the ulceration extending to the inferior maxillary bone, which was later found to be eroded. At the labiogingival junction there was a purulent exudate upon the mucous membrane. Before the animal was killed smears were taken from this exudate and the diseased areas, and cultures were made on all the ordinary culture media.

Postmortem examination of the animal showed the internal organs to be in a normal well-nourished condition, with the exception of a small area of verminous pneumonia of the upper lobe of the left lung. Further examination of the lip and mouth showed the diseased area to be a necrotic process, sharply circumscribed on the lower lip and adjacent soft parts, that extended from nearly the angles of the mouth toward the chin for about 1 cm. externally, while internally the whole mucous membrane of the lower lip and gum to the border of the incisor teeth was involved.

Section through this area showed the adjacent part of the lower maxillary bone to be eroded, and the softer tissues adjacent to the pale

necrotic area greatly reddened from congestion. Sections through the whole area were taken for histological study, with the following findings:

HISTOLOGICAL EXAMINATION. Sections stained with eosin-hematoxylin. The diseased area shows a gangrenous process involving all the tissues and extending from the skin surface of the lip to the gingival border.

All the cellular structures of the part show the same degree of degeneration (that is, loss of all protoplasmic and nuclear-staining characteristics) extending to an irregular but sharply circumscribed line of demarcation. Beyond this line of demarcation toward the healthy tissue there is a slight zone of inflammatory reaction, consisting chiefly of polymorphonuclear leukocytes and engorged and dilated capillary and larger bloodvessels. Beyond the reaction zone the normal healthy structures of the part are found.

Sections stained especially for bacteria show in the superficial parts, that is, the exposed part grossly, an infiltration with numerous forms of bacteria, in which cocci predominate and which are probably the ordinary mouth organisms. These infiltrating bacteria become less and less prominent as the deeper parts of the necrotic area are examined, and give way to bacillary and thread-like organisms which increase in number until a felt-like network of them is found near the line of demarcation. In this felt-like formation the bacillary organisms can be seen varying in thickness and length with the production of rods and threads extending in all directions.

Along the line of demarcation these organisms are less in number than in the felt-like area, but are still numerous and can be better studied here.

Here they vary in size and thickness, the more delicate threads predominating. Here also may be seen these organisms, some with angular bendings and some with irregularly stained bands, which seem to form small filaments which push between the cells of the part and the infiltrating leukocytes. These grow less in number and more delicate as they proceed toward the healthy tissue.

Sections stained by Gram's method are negative for these special bacillary and thread-like forms. A section stained by a special method, as recommended by Jensen for the Bacillus necroseos, which has been

found by others in spontaneous gangrenous processes in animals, failed to show the presence of these microörganisms.

Sections were also stained with Weigert's elastic stain to exclude possible confusion between the remnants of elastic tissue normal to the part and these threads, and showed that these threads were foreign to the tissue.

Bacteriological Examinations. The smears which were made from the exudate and the superficial necrotic areas were stained with Loeffler's alkaline methylene blue, and showed a great increase in the number of spiral microörganisms which were of various lengths and number of twists, some being robust and some very delicate resembling the spirillum of Vincent. In these spreads fusiform bacilli were also found, some of which were slightly curved; others showed irregular staining. Unfortunately no wet specimens were examined for motility.

The cultures which were grown aërobically showed the presence of streptococci and staphylococci, but no fusiform bacilli nor spirilla, particular attention being given to the growth of organisms in the water of condensation of the agar-agar and blood serum tubes.

No anaërobic cultures were made.

As a result of these findings and in view of the fact that Grenet[26] and Weaver and Tunnicliff had found the Bacillus fusiformis and spirillum of Vincent in normal mouths, and Angelici[27] had found the Bacillus fusiformis normally in man and lower animals, the question arose in regard to the frequency of spiral microörganisms and fusiform bacilli in the mouths of normal monkeys. To determine this smears were made from the mouths of 25 monkeys and examined for these organisms.

Of our 25 stained smears, 12 showed the presence of spiral microorganisms in very limited number; 18 showed the presence of fusiform bacilli also in limited number, and 11 showed both spiral and fusiform bacilli present in the same smear. In only 2 of the smears showing the spirals and fusiform bacilli in symbiosis did the spiral organism resemble the spirillum of Vincent. The monkey from which one of the latter smears was taken showed clinically an erosion of the mucous membrane of the lower lip, the monkey of the other smear being apparently normal. Wet specimens were made from 11 of these monkeys, but only in one instance was a spirillum found.

SUMMARY. 1. This is a case of noma occurring in a wild animal and showing the anatomical and clinical characteristics of the disease.

2. A fusiform bacillus and a spiral microörganism resembling Vincent's spirillum are found in the exudate and scrapings of the diseased area, there being also a great increase in spiral microorganisms.

3. The histological examination showed the presence of rod- and thread-like organisms similar to those described by Perthes, Brüning, and others in the diseased area, being especially located in the area of advancing disease.

4. A spiral organism resembling Vincent's spirillum in conjunction with a fusiform bacillus is found in the normal monkey's mouth.

5. Fusiform-shaped bacilli are frequently present in small numbers in the mouths of normal monkeys.

CONCLUSION. In conclusion we can make no claim that the organisms present are the cause of this condition of noma because of our failure to isolate and cultivate them, but would call attention to the fact that our findings are similar to those reported in a corresponding condition in human beings by others who have advanced the theory that they are the cause of noma or other gangrenous processes of the mouth.

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Address of the President.[1]

By JOSEPH McFARLAND, M.D.

As the time for our annual meeting drew nearer and nearer, marking the close of another year of our work as a society, I became more and more perplexed as to the best use to be made of that part of the evening set aside for the "President's Address." One after another a variety of subjects suggested themselves, but were cast aside with some impatience, for there was slowly but surely taking possession of my mind the conviction that upon this occasion the President of the Society ought to have a confidential talk with the members, reviewing the year's work critically, commenting favorably where conditions were good, adversely where otherwise, and forecasting the probabilities for the future.

I seemed to find stored away in the dark corners of memory's closet back records of certain addresses of my predecessors who pursued this same course, but lest I should become a repeater or an imitator I carefully avoided looking up any of their expressions. If, therefore, my remarks this evening follow the same general direction as theirs, it must be put down as a matter of some scientific interest that the same psychological necessity being experienced we were driven to the same general plan of action.

[1] Delivered at the Annual Meeting of the Pathological Society of Philadelphia, October 22, 1908.

Since our Society was formed, new conditions have arisen to hamper its success. When it was new there was probably no other association of medical men by whom medical subjects were treated chiefly from the scientific, *i. e.*, biological, point of view. In recent years, however, there have been formed the American Society of Bacteriologists, the Association of American Pathologists and Bacteriologists, the Laboratory Section of the American Public Health Association, and the Section on Pathology of the American Medical Association, in all of which bodies contributions are limited to technical papers in which the biological problems are dealt with to the exclusion of, or at the expense of, their immediate practical application.

In addition to these we have the Association of American Physicians with an omnivorous appetite and vast assimilative powers for papers upon the experimental investigation of all kinds of medical problems. All of these societies injure us, more or less directly, by drawing away many contributions that would otherwise fall to our portion, and indirectly by diminishing our relative importance. I might even include the Society of Normal and Pathological Physiology as aiding in this general disaffection.

But I must not make myself misunderstood. Though all of these organizations act more or less injuriously upon us as a Society, their formation is, in itself, of far-reaching good by drawing together capable workers from all parts of the country to read and discuss their best work and so promote national medical improvement. We are simply the small minority that always experiences some injustice when a great good is accomplished.

But, notwithstanding the stress of these losses, when one takes our program for a year and refreshes his recollection of what has really been accomplished, he cannot but be astonished at the extent and scope of our contributions and the variety of our exhibition material.

During the year beginning with the last annual election, October 10, 1907, and ending tonight, we have held fifteen meetings. There was no meeting November 28, Thanksgiving Day, none December 26, and none in June, July, August, September, or in the first two weeks of October, the late convention of our Society this autumn being caused by the meetings of the International Conference and Congress on Tuberculosis. The average attendance at these meetings was

small, but at such as might be designated "special occasions" the room overflowed.

During the year thirty-four papers were read. I suppose one would be justified in supposing that a critical survey of these papers would indicate the subjects of particular interest to the Philadelphia men, but allowances must be made for the presentation of much of the best work of Philadelphians before other and larger societies, and for the almost universal tendency to present some rare or curious thing rather than that of particular interest to the writer. This applies both to the papers read and to the specimens presented.

Some of the papers were devoted to the collection of known facts—useful summaries of the literature, and were usually presented by invitation. Some were descriptions and interpretations of things seen at the bed-side, at the autopsy table, or in the microscope. Some are records of interesting and ingenious experiments; and others are elaborate monographs upon special subjects. I have found it difficult to classify the papers otherwise than by subjects, and I may have done it badly. Alphabetically arranged they include the following:

Bacteriology and its Collaterals.

Diagnosis of Diphtheria by Smears.

Significance of Tubercle Bacilli in the Feces.

Actual Significance of Tubercle Bacilli in the Feces.

Bacterial Vaccines of Staphylococcic Strains; a Technique for their Preparation.

A Study of the Colon Aërogenes Group of Bacteria.

New and Improved Method for the Presumptive Test for Bacillus coli communis.

Bile Ducts.

Cysts of the Common Bile-duct.

Blood.

Study of the Hemopoietic Organs in Diphtheria and Tuberculosis.

Acute Lymphopenic Lymphatic Leukemia.

Proteolytic Ferments in a Case of Acute Leukemia.

Clinical.

Venous Pulse.
Observations on Urinary Tube Casts.

Experimental.

Liver Necroses from the Intravenous Injection of Ether during Life.
Volume and Specific Gravity of Organs removed at Autopsy.
Tissue Transplantation into Other Species.
The Coördination of Gastric and Intestinal Digestion by the Action of the Pyloric Sphincter.

Immunity.

Phagocytosis in Diphtheria.
Leukocyte Counts before and after the Administration of Antitoxin.

Kidneys—Nephritis.

Congenital Nephritis.

Nerves.

Interpretation of Appearances seen in a Peripheral Nerve.

Parasites.

Ciliated Organisms in the Liver of a Prairie Wolf.
Parasitic Nodular Conjunctivitis.
Cysticercus Tenuicollis.

Spleen.

Splenomegaly.

Syphilis.

The Present Status of the Spirocheta pallida (Treponema pallidum).
Immunity in Syphilis.

Tumors.

Production of Experimental Deciduoma.
Carcinoma of the Esophagus.
Metastatic Squamous Epithelioma of the Esophagus.
Sarcoma of the Eyelid.
Report of a Case of Metastatic Carcinoma of the Lung.
Brief Report of a Growth of the Testicle Resembling Sarcoma, with Metastases to the Lung.

Vessels.

Peri-arteritis Nodosa.

It appeals to me as remarkable and regrettable that there should have been no paper upon any subject related to chemistry, as the future of Pathology is supposed to lie hidden behind the chemical horizon. It is also striking that there should be complete silence upon subjects relating to the central nervous system, the cardiovascular system (we had only Longcope's paper upon "Peri-arteritis Nodosa"), the digestive system (we had only Lavenson's paper upon "Cysts of the Common Bile Duct"), the respiratory system, the bones and joints, the muscles, and the skin. Truly this is the passing of morbid anatomy! Has all been said upon these subjects, or do we fear that they are too familiar to be made the subjects of further communications? Anyone who has my experiences with recent graduates and young hospital residents, knows how little they know about hearts; and anyone who feels as uncertain as I do when I try to interpret what I see on the gastric mucosa, knows how much more he needs to know about the stomach.

But some of the omissions in writing have been made good by demonstration, and the table that was spread before us during the year groaned with good things enough to satisfy the most epicurean appetite for variety. Until I reviewed these lists, I had no idea, myself, how fertile was the soil we cultivate or how rich the harvest of our specimens.

Let us examine the lists and see what has been shown to us in a year. In all there were fifty-four specimens, and include the following:

Aneurysms:

One of the aorta.

Apparatus:

A new coverslip holder.

A guide for the use of the Maltwood's finder.

Arthritis:

Hypertrophic arthritis.

Bacteria:

Treponema pallidum.

A hitherto undescribed organism.

Bile Ducts:

Cysts of the common bile ducts.

Bladder:

Necrotic cystitis, with calcareous deposits.

Blood:

Specimens of leukemia.

Nucleated red crisis in leukemia.

Bones:

Skulls showing inadequate cranial repair.

Brain:

Abscess of the left ventricle.

Diffuse meningitis simulating abscess.

Cerebrospinal meningitis.

Calculi:

A tonsillolith.

Heart:

Aortic stenosis.

Aneurysm of the sinus of valsalva

Malignant endocarditis.

Endocarditis.

Bicuspid aortic valve.

Intestine:

Perforated ulcer.

Kidney:

Papillary cysts.

A pair of interesting kidneys.

Liver:

Cirrhosis.
Leukemia.
Angioma.
Fatty infiltration.
Necrosis after the intravenous injection of ether.
Metaplasia of the epithelial lining of the gall-bladder.
Section of gall-bladder containing a mass of lymphoid tissue resembling Peyer's patch.

Lung:

Infarction.
Cancer: Secondary to mamma.
Cancer: Secondary to mamma.
Rupture of subdiaphragmatic abscess into the lung.

Malformations:

Situs inversus vicerum sine dextrocardia.
Syndactylism.
Spina bifida.
Imperforate urethra.
Meckel's diverticulum.

Parasites:

Cysticercus tenuicollis.
Eustrongylus gigas.
Strongyloides intestinalis.
Strongyloides intestinalis.
Ciliated organism in the liver of a prairie wolf.
Parasitic organisms in nodular conjunctivitis.

Photographs:

Lumière photographs of various skin lesions.

Spleen:

Liver and spleen from leukemia.
Splenomegaly.

Stomach:

Carcinoma.
Squamous-cell carcinoma.

Syphilis:

Treponema pallidum.

Tuberculosis:

Of retroperitoneal glands.

Of seminal vesicles.

Tumors:

Specimen showing dissemination of a uterine carcinoma through the retroperitoneal glands and thoracic duct.

Carcinoma of stomach.

Squamous-cell carcinoma of stomach.

Adrenal "rests"—one developing into a hypernephroma.

Syncytioma.

Angioma of the liver.

Malignant struma: Microscopic specimen from Prof. Langhans.

Cancer of lung.

Cancer of lung.

Syncytioma of the testes.

Giant-cell sarcoma of the forearm.

Sarcoma of the glands of the esophagus.

Sarcoma of the eyelid.

Vessels:

Aneurysms of the aorta.

Peri-arteritis nodosa.

It seems to me that this list is one upon which we can congratulate ourselves. It is very interesting, and I think important to notice in passing that these specimens were presented by no less than thirty-six different individuals; and when to this fact is added that sixty-five *different persons*, or 22 per cent. of our entire membership, participated in the meetings of the year, otherwise than by discussion, I think it is very gratifying and shows that interest in subjects related to pathology is not confined to a few men who happen to teach the specialty, but extends to many others whose chief activities are in hospitals and dispensaries.

Considerable profit and enjoyment resulted from the excellent plan of the Business Committee, upon the efficiency of whose work I cannot too favorably comment, in arranging the symposium upon "Syphilis," at which Drs. Flexner and Torry gave a beautiful demonstration of living spirochetæ; and the symposium upon the "Para-

thyroids," at which we were profitably entertained by Professor Halsted and Dr. W. G. MacCallum.

We all remember with interest the address of Professor R. M. Pearce, at the semi-annual Conversational Meeting upon "The Theory of Chemical Correlation as Applied to the Pathology of the Kidney," and the two other addresses by invited guests, Professor W. B. Cannon, of Harvard, upon "The Coördination of Gastric and Intestinal Digestion by the Action of the Pyloric Sphincter," and of our own Dr. Donaldson upon the "Interpretation of Appearances Seen in a Peripheral Nerve."

I am pleased to announce that our membership numbers three hundred and two; during the year we lost six members but gained nineteen, so that we are richer than before.

It is with profound regret that I recall our loss in the death of Dr. J. Dutton Steele. The patience and perseverance with which he applied himself to the study of those problems that interested him should be an inspiration to every member of our Society.

We are about to embark upon another year's endeavor. With this excellent record behind us, we can confidently hope for a prosperous future.

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PROCEEDINGS

OF THE

PATHOLOGICAL SOCIETY

OF PHILADELPHIA.

NEW SERIES, VOLUME XI.
OLD SERIES, VOLUME XXIX.

CONTAINING THE TRANSACTIONS OF THE SOCIETY FROM OCTOBER, 1907, TO OCTOBER, 1908.

EDITED BY

FRED H. KLAER, M.D.,

RECORDER OF THE SOCIETY.

PHILADELPHIA:
PRINTED FOR THE SOCIETY BY WM. J. DORNAN.
1908.

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1905 Klaer, F. H., 334 South Sixteenth Street.
1900 Klapp, W. P., 1716 Spruce Street.
1898 Knipe, J. C., 2035 Chestnut Street.
1901 Kohn, B., 1325 North Thirteenth Street.
1908 Kotz, Adam L., Easton, Pa.
1908 Krumbhaar, Ed. B., Pennsylvania Hospital.
1895 Krusen, Wilmer, 127 North Twentieth Street.
1892 Kyle, D. Braden, 1517 Walnut Street.

1899 Landis, H. R. M., 130 South Twenty-third Street.
1890 Laplace, Ernest, 1828 South Rittenhouse Square.
1905 Lavenson, Ralph S., 1218 Locust Street.
1894 Leach, W. W., Eastern State Penitentiary, Medical Department.

ELECTED

1869 Leaman, Henry, 828 North Broad Street.
1888 Leidy, Joseph, 1319 Locust Street.
1905 L'Engle, Edward M., 132 South Twenty-third Street.
1887 Leopold, Isaac, 1520 Franklin Street.
1904 Leopold, S., 1632 Franklin Street.
1875 Lewis, Morris J., 1316 Locust Street.
1894 Lincoln, C. W., 314 E. Lancaster Avenue, St. David's, Pa.
1899 Lindauer, Eugene, 2018 North Thirty-second Street.
1886 Lloyd, James Hendrie, 3918 Walnut Street.
1898 Lodholtz, Edward, 3103 Diamond Street.
1903 Loeb, Leo, University of Pennsylvania.
1894 Loeb, Ludwig, 1421 North Fifteenth Street.
1901 Longcope, W. T., 323 South Sixteenth Street.
1875 Longstreth, Morris, 1416 Spruce Street.
Ludlum, S. D., Friends' Asylum, Frankford.
1903 Lukens, G. T., Fifth Ave. and Fayette St., Conshohocken, Pa.

1896 McCarthy, D. J., 1329 Spruce Street.
1904 McClary, Samuel, 3d, 308 South Fifty-second Street.
1880 McClellan, George, 1116 Spruce Street.
1892 McFarland, J., 442 W. Stafford Street, Germantown.
1902 McGowan, J. M., 406 South Broad Street.
1894 McKee, James H., 1519 Poplar Street.
1906 McLaughlin, J. J., 1813 South Broad Street.
1905 Maier, E. G., 2242 North Broad Street.
1905 Maier, F. Hurst, 2244 North Broad Street and 1900 Chestnut St.
1902 Marshall, C. J., 2004 Pine Street.
1899 Masland, H. C., 2134 North Nineteenth Street.
1873 Meigs, Arthur V., 1322 Walnut Street.
1906 Meyers, Milton K., 2134 North Eighteenth Street.
1878 Mills, C. K., 1909 Chestnut Street.
1906 Mitchell, Charlotte B., 1707 Pine Street.
1884 Mitchell, J. K., 1730 Spruce Street.
1907 Montgomery, C. M., 256 South Fifteenth Street.
1898 Morris, Henry, 313 South Sixteenth Street.
1885 Morrison, William H., 8021 Frankford Avenue, Holmesburg, Pa.
1887 Morton, S. W., 1933 Chestnut Street.
1899 Müller, G. P., 334 South Fifteenth Street.
1906 Muschlitz, Chas. H., 3611 Spruce Street.
1880 Musser, J. H., 1927 Chestnut Street.

1879 Neff, J. S., Cynwyd, Montgomery Co., Pa.
1900 Newlin, A., 253 South Thirteenth Street.

ELECTED

1898 Newton, R. D., 6137 Vine Street.
1902 Noble, C. P., 1509 Locust Street.
1900 Norris, Geo. W., 1530 Locust Street.

1894 O'Malley, Joseph, 2228 South Broad Street.
1900 O'Reilley, Charles A., 127 South Eighteenth Street.

1893 Packard, F. R., 1836 Pine Street.
1907 Pancoast, H. K., 4238 Pine Street.
1901 Patterson, F. D., 2103 Locust Street.
1900 Pearson, L., Veterinary Department, University of Pennsylvania.
1903 Pemberton, R., 1953 Locust Street.
1897 Pepper, William, 1811 Spruce Street.
1895 Perkins, F. M., 1428 Pine Street.
1897 Peter, L. C., 1700 Oxford Street.
1902 Pfahler, G. E., 1321 Spruce Street.
1885 Piersol, G. A., 4724 Chester Avenue.
1905 Piersol, Geo. M., 344 South Sixteenth Street.
1907 Pitfield, R. L., 5211 Wayne Avenue.
1890 Potts, C. S., 1733 Chestnut Street.
1908 Prime, Frederick, Jr., 344 South Sixteenth Street.
1901 Purves, G. M., 4204 Walnut Street.

1905 Radasch, H. E., 914 South Forty-seventh Street.
1904 Rahte, Walter E., 309 South Sixteenth Street.
1885 Randall, B. A., 1717 Locust Street.
1904 Reber, Wendell, 1212 Spruce Street.
1894 Reckefus, C. H., 506 North Sixth Street.
1906 Reichel, John, William Pepper Laboratory.
1907 Repplier, S. J., 328 South Sixteenth Street.
1894 Rhein, J. H. W., 1732 Pine Street.
1903 Rhein, R. D., 2016 Pine Street.
1907 Richardson, W. W., State Hospital for Insane, Norristown, Pa.
1894 Riesman, David, 1715 Spruce Street.
1891 Ring, G. O., 2012 Chestnut Street.
1904 Rivas, Damaso, corner Sixty-seventh and Vine Streets.
1876 Roberts, John B., 313 South Seventeenth Street.
1884 Robertson, W. E., 320 South Sixteenth Street.
1901 Robinson, E. T., 1326 Pine Street.
1904 Robinson, G. Canby, Pennsylvania Hospital.
1889 Robinson, William Duffield, 2012 Mount Vernon Street.
1908 Rodman, J. Stewart, 1904 Chestnut Street.
1901 Roe, W. J., 1210 Locust Street.

ELECTED

1898 Rosenberger, R. C., 2330 North Thirteenth Street.
1894 Ross, George, 1721 Spruce Street.
1900 Roussel, A. E., 2112 Pine Street.
1905 Royer, Franklin B., Municipal Hospital.

1895 Sailer, Joseph, 248 South Twenty-first Street.
1905 Salus, H. W., 1118 Pine Street.
1904 Sargent, A. Alonzo, 939 Spruce Street.
1890 Sartain, Paul J., 212 West Logan Square.
1895 Schamberg, J. F., 1922 Spruce Street.
1904 Schumann, E. A., 15 Pelham Road.
1882 de Schweinitz, G. E., 1705 Walnut Street.
1893 Scott, J. A., 1834 Pine Street.
1907 Sharpless, F. C., Rosemont, Pa.
1898 Sharpless, Wm. T., 100 South Church Street, West Chester, Pa.
1902 Shields, W. G., Jr., 412 West School House Lane, Germantown.
1905 Shoemaker, Harlan, 1618 Spruce Street.
1889 Shoemaker, Harvey, 2011 Chestnut Street.
1896 Shumway, E. A., 2007 Chestnut Street.
1899 Sinclair, J. F., 4103 Walnut Street.
1902 Sinkler, Francis W., 220 South Sixteenth Street.
1868 Sinkler, Wharton, 1606 Walnut Street.
1901 Siter, E. H., 2038 Locust Street.
1881 Skillern, P. G., 241 South Thirteenth Street.
1903 Small, J. H., 914 South Forty-eighth Street.
1903 Smith, A. J., Medical Department, University of Pennsylvania.
1906 Somers, Henry J., State Hospital for Insane, Norristown, Pa.
1902 Somers, Lewis S., 3554 North Broad Street.
1905 Speese, John, 328 South Sixteenth Street.
1893 Spellissey, Joseph M., 110 South Eighteenth Street.
1896 Spiller, William G., 4409 Pine Street.
1904 St. John, E. Q., 1833 Chestnut Street.
1890 Stahl, B. F., 1727 Pine Street.
1901 Stanton, W. B., 732 Pine Street.
1904 Stellwagon, Thomas C., Jr., 1121 Spruce Street.
1889 Stengel, Alfred, 1811 Spruce Street.
1889 Stevens, Arthur A., 314 South Sixteenth Street.
1896 Stewart, Alonzo H., 252 North Twelfth Street.
1899 Stout, G. C., 1611 Walnut Street.
1905 Stout, Philip S., 4625 Woodland Avenue.
1884 Strittmatter, I. P., 999 North Sixth Street.
1869 Stryker, Samuel S., 3833 Walnut Street.
1894 Swan, John M., 3713 Walnut Street.

ELECTED

1899 Talley, J. E., 1927 Chestnut Street.
1897 Teller, Wm. H., 1713 Green Street.
1906 Thomas, B. A., 1819 Chestnut Street.
1894 Thomas, W. Hersey, 1421 North Seventeenth Street
1902 Thornton, E. Q., 1331 Pine Street.
1902 Tracy, S. E., 1415 Walnut Street.
1897 Tucker, Henry, 2000 Pine Street.
1863 Tyson, James, 1506 Spruce Street.
1890 Tyson, T. Mellor, 1506 Spruce Street.

1900 Uhle, A. A., 1327 Jefferson Street.
1904 Ullom, J. T., 24 Carpenter Street, Germantown.

1894 Vandervoort, C. A., 3311 North Broad Street.
1905 Van Kaathoven, J. J. A., 1715 Spruce Street.

1903 Walker, J. K., 1632 Spruce Street.
1891 Wallace, James, 1921 Chestnut Street.
1899 Walsh, J., 732 Pine Street.
1890 Warder, C. B., 1633 Spruce Street.
1903 Weisenburg, T. H., 2030 Chestnut Street.
1887 Westcott, T. S., 1833 Spruce Street.
1908 Weston, Paul G., 1632 Green Street.
1897 White, Courtland Y., 1808 Diamond Street.
1873 White, J. William, 1810 South Rittenhouse Square.
1901 Whiteway, Harold M., 1924 Chestnut Street.
1893 Whiting, A. D., 1523 Spruce Street.
1905 Wieder, Henry S., 2131 North Fifteenth Street.
1868 Willard, De Forest, 1901 Chestnut Street.
1898 Willson, R. N., 1708 Locust Street.
1906 Wilson, J. D., Jefferson Medical College.
1869 Wilson, James C., 1509 Walnut Street.
1907 Wilson, Oscar H., 5128 Spruce Street.
1891 Wilson, Samuel M., 1517 Arch Street.
1901 Winsor, Henry, Manilla, P. I.
1900 Wister, J. W., 5430 Germantown Avenue.
1907 Wolf, Henry F., 1804 Green Street.
1890 Wood, A. C., 128 South Seventeenth Street.
1898 Wood, George B., 129 South Eighteenth Street.
1906 Wood, Harold B., 5038 Pine Street.
1865 Woods, D. Flavel, 1501 Spruce Street.
1899 Worden, C. B., 322 South Sixteenth Street.

1902 Zimlick, A. J., 702 East Chelten Avenue.
1895 Zimmerman, Mason W., 1522 Locust Street.

NON-RESIDENT MEMBERS.

*Subscribers to Proceedings.

Alburger, Henry R., Bloomington Indiana.
Ball, M. V., Warren, Pa.
Barnes, A. S., Jr., Missouri Trust Building, St. Louis, Mo.
*Biggs, M. H., Rutherfordton, N. C.
Carter, W. S., Galveston, Texas.
Coca, A. F., University of Pennsylvania.
*Dock, George, 602 East Huron Street, Ann Arbor, Mich.
Edwards, W. A., San Diego, Cal.
Egbert, Joseph P., Wayne, Delaware County, Pa.
Gaylord, H. R., Buffalo, N. Y.
Gerson, T. P., 1621 Ingraham Street, Los Angeles, Cal.
Hamaker, W. D., Meadville, Pa.
*Hamann, C. A., 404 Osborn Building, Cleveland, Ohio.
Harris, H. F., Atlanta, Georgia.
Harrison, W. H., Harrisburg, Pa.
Hatch, John L., 2010 Fifth Avenue, New York City.
Hickman, W. A., 612 Pacific Avenue, Atlantic City.
Holder, C. A., Colorado Springs, Col.
Howard, F. H., Williamstown, Massachusetts.
Hunt, J. R., 102 East Fifty-seventh Street, New York.
Jamar, John H., Elkton, Md.
Kiefer, Charles, United States Army.
Kinyoun, J. J., Glenolden, Pa.
Lincoln, W. R., Cleveland, Ohio.
*McConnell, G., 4175 Washington Avenue, St. Louis, Mo.
de Nancrède, C. B. G., Ann Arbor, Mich.
*Pearce, R. M., Bender Hygienic Laboratory, Albany, N. Y.
Pease, H. D., Buffalo, N. Y.

Powell, W. M., 122 West Minor Street, West Chester, Pa.
*Rahter, C. A. 110 North Second Street, Harrisburg, Pa.
*Ravenel, M. P., University of Wisconsin, Madison, Wisc.
Reynolds, W., 27 South Indiana Avenue, Atlantic City.
Slifer, Henry F., North Wales, Pa.
Stadelman, Eugene, "Magistral," Maria Del Oro, Dgo., Mex.
Stubbs, R. P., Wilmington, Delaware.
Taylor, L. H., Wilkesbarre, Pa.
Toulmin, H., Haverford, Pa.
Wells, G. M., Wayne, Delaware County, Pa.
Wetherill, R. B., Lafayette, Ind.
Williams, H. L., Minneapolis, Minnesota.
Wilmarth, A. W., State Home for the Feeble-minded, Chippewa Falls, Wisconsin.

CORRESPONDING MEMBERS.

ELECTED

1885 Dent, Clinton T., Assistant Surgeon and Lecturer on Practical Surgery at St. George's Hospital, Surgeon to Belgrave Hospital for Children, London, England.

1888 Fedeli, Gregorio, Rome, Italy.

1908 Flexner, Simon, Rockefeller Institute, New York City.

1890 Gibbs, Heneage, 585 John R. Street, Detroit, Michigan.

1908 Novy, H. G., Ann Arbor, Michigan.

1908 Osler, William, University of Oxford, England.

1886 Pye-Smith, P. H., Guy's Hospital, London, England.

1908 Taylor, A. E., University of California, San Francisco, Cal.

1898 Welch, William H., Professor of Pathology, Johns Hopkins University, Baltimore, Maryland.